Effect of respiration on venous return and stroke volume in cardiac tamponade

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*Measuremants have been made of pressure and blood velocity in venae cavae and aorta in a
patient with severe cardiac tamponade in whom there was pulsus paradoxus. The characteristic
pressure changes of pulsus paradoxus were associated with variations in peak blood velocity in
the ascending aorta and stroke output. Maximum filling of the right side of the heart occurred
during inspiration, and was associated in time with minimum left ventricular stroke volume.*

*These findings point to competition for space by the ventricles in the distended pericardial
sac as being the major factor in the production of pulsus paradoxus.*

Pulsus paradoxus has long been recognized and was defined by Gauchat and Katz (1924) as a 'rhythmical pulse which diminishes perceptibly in amplitude or is totally obliterated during inspiration in all palpable arteries.' At the same time they described two groups of cases in which pulsus paradoxus occurred, those with enhanced intrathoracic pressure variation and those with cardiac tamponade. These authors also believed (Katz and Gauchat, 1924) that in cases of tamponade when the intrapericardial pressure was high throughout the respiratory cycle the pulmonary veins increased in size on inspiration, with consequent transient pooling of blood and reduction in the cardiac filling and left ventricular stroke volume. Dock (1961), on the other hand, suggested that in cardiac tamponade pericardial pressure rose during inspiration as a result of traction on the pericardium. Dornhorst, Howard, and Leathart (1952a), supported experimentally by Shabetai *et al.* (1965), considered that in cardiac tamponade there was competition by the two ventricles for a fixed total diastolic volume; the increased right ventricular filling in inspiration results in decreased left-sided filling. Guntheroth, Morgan, and Mullins (1967) have made observations on the blood flow and pressure in experimental tamponade in dogs, and have concluded that the variations in blood pressure during tamponade are the result of respiratory effects on the

right ventricular stroke volume, delayed by transit through the pulmonary bed and exaggerated by a small left ventricular stroke volume in a vasoconstricted state. The recent introduction of a miniature catheter tip electromagnetic velocity probe that can be used at the time of routine cardiac catheterization (Mills and Shillingford, 1967) has enabled us to study in detail the instantaneous pressure flow relation in a case of cardiac tamponade.

Methods

A 24-year-old man, three years before the present investigation at the National Institutes of Health, Bethesda, had developed massive mediastinal adenopathy. Biopsy of a cervical lymph node had confirmed the diagnosis of Hodgkin's disease. He was subsequently treated with radiotherapy to the chest and neck. He became asymptomatic with complete regression of all palpable adenopathy. Six months before admission the cardiothoracic ratio slowly increased and there was a change in the heart contour to a globular shape. Two weeks before admission he developed dyspnoea and orthopnoea. There was pulsus paradoxus. A chest *x*-ray showed a greatly enlarged globular cardiac outline and a right pleural effusion. During cardiac catheterization 1800 ml. of sero-sanguineous pericardial fluid was removed, with subjective relief and return of vascular and intracardiac pressures to normal limits. Subsequently there was reaccumulation of the pericardial effusion and he later underwent pericardectomy. Since then his condition has been good and he has been free from cardiovascular and respiratory symptoms.

Procedure during cardiac catheterization
Left and right routine cardiac catheterization
was performed with the use of a catheter incor-
porating an electromagnetic velocity probe at its
tip* (Mills and Shillingford, 1967) together with
a lumen for the simultaneous measurement of
pressure. The pressure transducer employed was
a Statham P23Gb strain gauge, and recordings
were made on an Electronics for Medicine photo-
graphic recorder. The natural resonant frequency
of the pressure recording system was 45 c/s with
a damping ratio of 0·19. The frequency response
of the electromagnetic velocity probe system was
± 1 per cent to 10 c/s, with a delay of 5·9/msec. Res-
piratory movements were recorded from pressure
changes within an air-filled compliant tube en-
circling the chest. Brachial artery pressure was
monitored with a P23Db strain gauge.

Recordings of pressure and velocity were made
in the superior and inferior venae cavae and in
the ascending aorta. Unfortunately, attempts to
obtain satisfactory blood velocity records in the
pulmonary artery were unsuccessful. The catheter
was seen on fluoroscopy to be moving vigorously
during each cardiac cycle while it was in the
pulmonary artery and the velocity record was
greatly disturbed as a result.

Cardiac output was measured by the indicator
dilution method, using a Gilford densitometer
and indocyanine green. The diameter of the
aorta was determined by biplane cine-angiocardio-
graphy during the aortic injection of 45 per cent
Hypaque.

Pressure and blood velocity were recorded in
the superior and inferior venae cavae and in the
ascending aorta before and after removal of
1800 ml. of pericardial fluid.

Results

The patient's heart rate averaged 111/min.
before pericardiocentesis, while the pres-
sures in the intracardiac chambers and main
vessels were: mean right atrial pressure
18 mm. Hg, right ventricular pressure 36/22
mm. Hg, pulmonary artery pressure 36/20
mm. Hg, mean pulmonary artery wedge pres-
sure 22 mm. Hg, and ascending aortic pres-
sure 120/80–70/66 mm. Hg. The cardiac
output was 3·4 l./min. (cardiac index 1·9
l. min.$^{-1}$m.$^{-2}$). The cross-sectional area of
the aorta, as measured by angiocardiography,
was 4·52 cm.^{2} After removal of 1800 ml.
pericardial fluid the heart rate was 98/min.
and the pressures were as follows: mean right
atrial pressure 5 mm. Hg, right ventricular
pressure 24/6 mm. Hg, pulmonary artery
pressure 24/8 mm. Hg, mean pulmonary
artery wedge pressure 4 mm. Hg, and ascend-
ing aortic pressure 120/70 mm. Hg. The
cardiac output was 6·4 l./min. (cardiac index
3·6 l. min.$^{-1}$m.$^{-2}$).

* Now manufactured by S.E. Laboratories, Feltham,
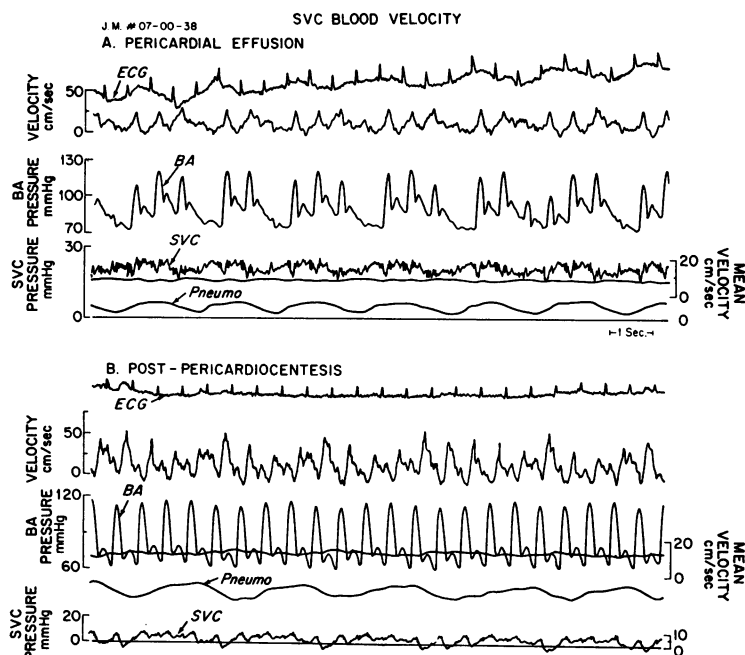Middx., England.

FIG. 1 *From above downwards, simultan-
eous recording of electrocardiogram (ECG),
superior vena caval blood velocity, brachial
arterial (BA) pressure, superior vena caval
(SVC) pressure, and mean velocity before
(A) and after (B) pericardiocentesis. In this
and the other Figures inspiration is shown by
a downward deflexion of the pneumogram
(pneumo).*

**Superior and inferior vena caval flows
and pressures** Fig. 1 shows a simultaneous
recording of the electrocardiogram, superior
vena caval blood velocity and pressures,
brachial arterial pressure, and pneumogram
before and after pericardiocentesis. The areas
below the superior vena caval blood velocity
tracing, between successive R waves of the
electrocardiogram and above the zero velocity
baseline, should be proportional to the superior
vena caval stroke volume, that is, the volume
delivered through the superior vena cava in
each cardiac cycle, if the assumption is made
that the vessel size changes little. Such areas
over three respiratory cycles, before peri-
cardiocentesis, were measured, and are
plotted in Fig. 2, each being expressed rela-
tive to the first; also plotted are the brachial
arterial pulse pressure, systolic pressure, and
the pneumogram. It will be seen that there
is a variation in the vena caval flow and bra-
chial arterial pressure during the respiratory
cycle. In an expiratory pause, however, the
brachial arterial pressure fell only slightly
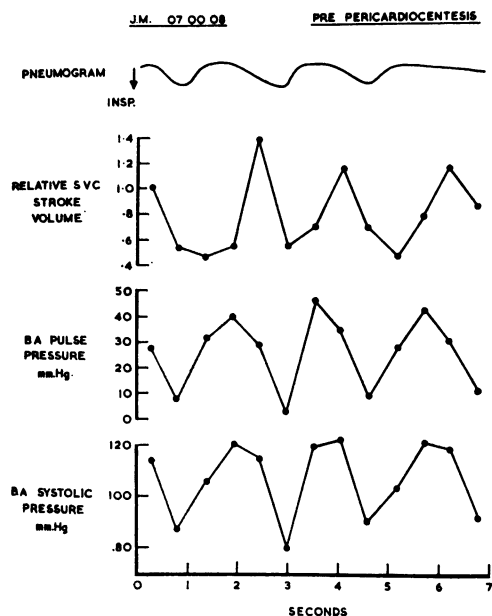before the steep decline at the next inspira-

FIG. 2 *Relative superior vena caval (SVC) stroke volumes (volumes delivered through the SVC in each cardiac cycle), brachial arterial (BA) pulse pressure, brachial arterial systolic pressure, and pneumogram in the patient with a pericardial effusion.*

FIG. 3 *Simultaneous recording of the electrocardiogram, superior vena caval (SVC) blood velocity, brachial blood pressure, and pneumogram before pericardiocentesis. Note that during the expiratory pause there is little change in brachial artery pressure until the next inspiration.*

tion (Fig. 3). The maximum superior vena caval blood velocity occurs during ventricular systole, and its magnitude varies with respiration, being largest during early inspiration phase and smallest during early expiration. The largest superior vena caval stroke volume occurs during early inspiration, one beat before the smallest pulse pressure detected in the brachial artery. It is likely that the largest right ventricular stroke volume occurs in the cycle following the largest superior vena caval stroke inflow volume.

Aortic pressure and flow   Fig. 4 shows the relation between ascending aortic blood velocity, pressure, and the pneumogram. The changes in relative left ventricular stroke volume, measured by planimetry over three respiratory cycles and expressed relative to the first value, are shown in Fig. 5; also shown are the aortic pressure and pulse pressure. The characteristic features in the pressure record of marked pulsus paradoxus are clear; there are similar changes in relative left ventricular stroke volume, which is largest during end-expiration. On expiration the aortic blood velocity rose to as high as 80 cm./sec. and on inspiration there was often little or no left ventricular ejection.

After removal of the pericardial effusion there were only minimal variations in pulse pressure and peak blood velocity in the ascending aorta.

Intrapericardial pressure and haemodynamics   Fig. 6 shows tracings of intrapericardial pressures, measured during pericardiocentesis and brachial arterial blood
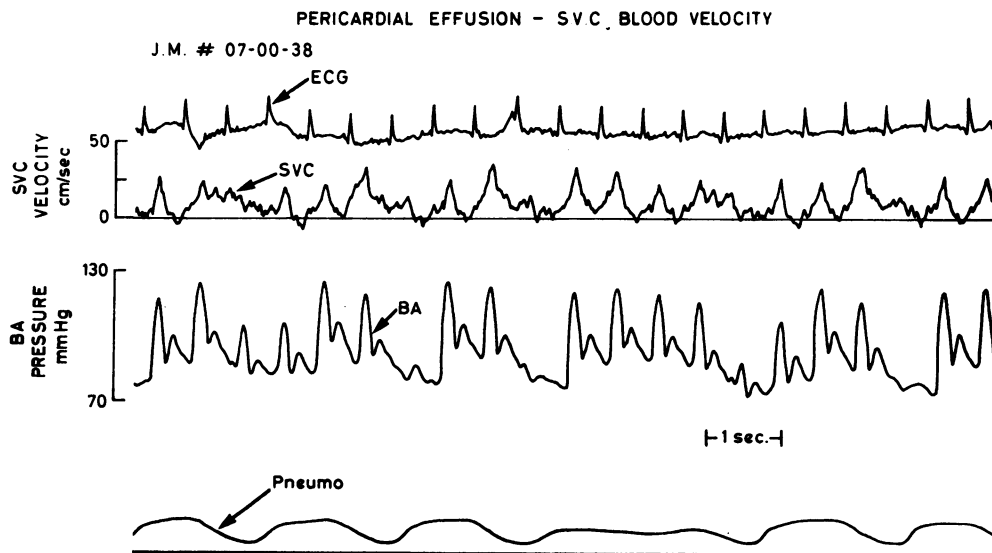
pressure. During systole there is a sudden fall in intrapericardial pressure. This fall is similar in magnitude when the brachial arterial pulse pressure is large or small, suggesting that the volume of blood ejected by the heart in the early part of systole is constant throughout the respiratory cycle, with ejection from the left ventricle smallest when that from the right ventricle is largest. After removal of 1800 ml. pericardial fluid the mean intrapericardial pressure fell from its original value of 18 mm. Hg to −2 mm. Hg.

Discussion

In the normal human subject arterial systolic pressure falls slightly during inspiration. That the act of inspiration is not itself directly responsible for this fall is suggested by the fact that at slow rates of respiration the fall occurs during expiration (Dornhorst, Howard, and Leathart, 1952b). Experimental work in animals has shown (Hoffman *et al.*, 1965; Charlier, 1968) that the larger right ventricular stroke volumes produced during inspiration are delayed in transit through the pulmonary bed, arriving at the left side of the heart two to three beats later; and similar conclusions were reached by Goldblatt *et al.* (1963), who studied the effects of respiration on right and left ventricular size in human subjects.

On physical grounds a similar delay in the transit of blood across the lungs must occur in pulsus paradoxus, but it appears that delay is not the dominant mechanism for the respiratory variations of the pulse then observed. Our findings are consistent with the hypothesis put forward by Dornhorst *et al.* (1952a) who explained the mechanism of the changes in cardiac tamponade as follows: 'normally the inspiratory drop in intrathoracic pressure is equally applied to the left ventricle and pulmonary veins. No change in left ventricular effective pressure ensues and no material immediate change in filling or ejection. Right ventricular effective filling pressure does increase, because the systematic veins are largely extrathoracic. Thus increased filling of the right ventricle is without effect on the left. When the pericardium is distended the effect is different. In that case increased filling of one ventricle will increase the intrapericardial pressure and hence tend to hinder filling of the other. With inspiration the intrapericardial pressure starts to fall, but does not fall as far as does the intrathoracic pressure because of increased right ventricular filling. Decreased filling of the left ventricle thus occurs.'
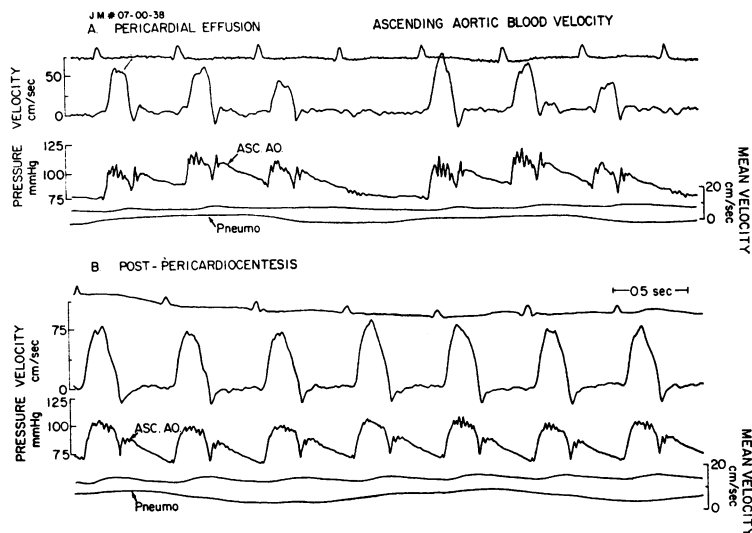

FIG. 4 *Simultaneous recording of electro-cardiogram, ascending aortic (ASC. AO.) blood velocity and pressure, and pneumogram before (A) and after (B) pericardiocentensis.*

Support for this hypothesis is given by the fact that the left ventricular stroke volume decreased simultaneously with the large right ventricular stroke volume and that during an expiratory pause there was little fall in blood

FIG. 5 *Relative left ventricular (LV) stroke volume, aortic pulse pressure, aortic systolic pressure, and pneumogram during the respiratory cycle in the patient with a pericardial effusion.*
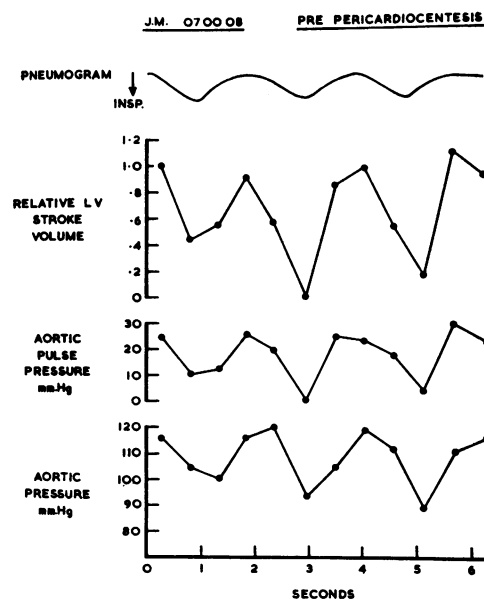
pressure, this only decreasing during the next inspiration. It could be argued that the fall in left-sided stroke volume on inspiration is the result of increased pulmonary venous volume (Golinko, Kaplan, and Rudolph, 1963). It is likely that this is to some extent true, but Shabetai *et al.* (1965) showed that in apnoeic animals a sudden increase of venous return to the right atrium was immediately followed by a decrease in aortic pressure. This important experimental finding argues against the hypothesis of Guntheroth *et al.* (1967), who attributed variation in right ventricular stroke volume largely to transit delay through the pulmonary bed. However, it supports the theory that competition between the ventricles for space within the distended pericardial sac is more important than pulmonary venous pooling as a mechanism for the production of pulsus paradoxus.
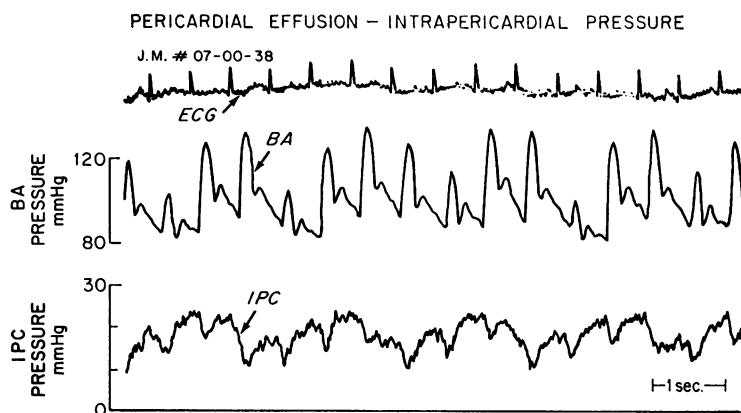

FIG. 6   *Simultaneous recording of the electrocardiogram, brachial arterial pressure, and intrapericardial (IPC) pressure in the presence of pericardial tamponade.*

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